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# Micro Rnas' Roles in Coronary Artery Disease and Revascularization

**Graciela Carlos***

Division of Medical Engineering Research, National Health Research Institutes, Miaoli, 350, Taiwan

**Corresponding author:** Graciela Carlos

Graciela69@gmail.com

Division of Medical Engineering Research, National Health Research Institutes, Miaoli, 350, Taiwan

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## Abstract

The vascular wall's chronic inflammatory response is commonly regarded as atherosclerosis, and its complications contribute significantly to patient mortality. Patients with atherosclerotic disease frequently undergo an angioplasty with stent replacement. However, angioplasty patients typically experience a high rate of restenosis. New signalling molecules that regulate the progression of atherosclerosis and restenosis have continuously been discovered, despite the well-established pathophysiological mechanisms that underlie these conditions. A new class of gene regulators known as microRNAs (miRs) function through translational inhibition or activation and transcriptional degradation

More than 30% of the cell's genes can be directly controlled by miRs. As a result, it is accepted that miRs play a crucial role in normal development, physiology, and pathogenesis. Diverse vascular diseases have been linked to variations in miR expression profiles. It has been discovered that miRs regulate a wide range of vascular cell functions, including cell differentiation, contraction, migration, proliferation, and inflammation. These functions are also involved in angiogenesis, the formation of noontime, and the lipid metabolism that underlies a variety of vascular diseases. Atherosclerosis and restenosis are characterized by the regulation of vascular cell function by miRs, which are the subject of this review. Clinical diagnostic and therapeutic approaches for vascular diseases caused by atherosclerosis and restenosis will likely benefit from these discoveries

**Keywords:** Cardiovascular; Syndromes; Surgical treatments

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## Introduction

A number of cardiovascular-related diseases are brought on by atherosclerosis, a chronic and progressive pathology marked by the accumulation of lipid and fibrous components in the large arteries. Atherosclerosis has a colossal effect in creating and created nations, addressing the fundamental reason for roughly half of passing. Over the past century, our understanding of this important disease's pathophysiology has improved. Vascular endothelial cells (ECs) become dysfunctional as a result of chemical influences, such as cytokines and growth factors [1], and hemodynamic forces [2], the pathogenic feature of atherosclerosis, according to extensive evidence. Leukocytes and monocytes are recruited to bind to the endothelium and migrate into the vessel wall by activated ECs with high levels of expression of various leukocyte adhesion molecules. After that, the lesion goes through the following stages: foam cell growth, the accumulation of fatty streaks, the migration and proliferation of vascular smooth muscle cells (VSMCs), and the formation of a fibrous cap. Lastly, thrombosis in advanced lesions-related complications that result in unstable coronary syndromes, myocardial infarction, and stroke is caused by the unstable fibrous cap's rupture. New approaches to treatment and prevention are made possible by the understanding that atherosclerosis is a vascular pathology brought on by an inflammatory response. In clinical trials, immune suppressants and anti-inflammatory agents might be used. However, percutaneous trans-luminal coronary angioplasty (PTCA) and stent placement remain the most common surgical treatments for patients with atherosclerosis.

## Discussion

In order to increase the inner diameter of the artery in various vascular locations, angioplasty and the placement of stents remove the occlusion. The hemodynamic flow rate is only improved by these treatments, which results in normal blood flow. Restenosis is a persistent complication that occurs 30–40% of the time three to six months after treatment, despite the fact that these treatments have been utilized in a large number of patients with atherosclerotic disease over the past few decades. Restenosis is actually a vascular injury caused by balloon dilation and stent replacement during angioplasty [4], despite the fact that restenosis and atherosclerosis are recognized as inflammatory processes that occur in response to injury [3]. Restenosis is distinct from atherosclerosis in terms of pathophysiology. Neointimal hyperplasia, extracellular matrix remodeling, and VSMC proliferation and migration all show these distinctions. An increased incidence of restenosis following angioplasty is linked to clinical and anatomical variables [5]. A brand-new class of gene regulators, microRNAs (miRs) are newly discovered endogenous, noncoding, single-stranded RNAs with 18–22 nucleotides. In 1993, during the development of Caenorhabditis elegans, the first miR, lin-4, was discovered [6 ] came up with an integrative strategy that used bioinformatic prediction, microarray analysis, and sequence-directed cloning to discover that there are over 800 miRs in humans. Over 15,000 miR gene loci have been found in over 140 species, and the miRBase16 contains over 17,000 distinct mature miR sequences [8]. In the 3'-untranslated regions (3'-UTRs), where MiRs bind to their target genes, messenger RNA (mRNA) is either directly degraded or translationally repressed by a perfect or imperfect complement. This suggests that miRs can control the expression of tens of thousands or hundreds of genes. As a result, it should not come as a surprise that miRs are involved in controlling all major cellular functions [9].

Over the past few decades, it has been well established that angioplasty-induced vascular pathologies like atherosclerosis, hypertension, coronary artery disease, and restenosis are caused by pathophysiological mechanisms. These vascular pathologies and diseases are caused by vascular properties like angiogenesis, re-endothelialization, and the formation of neointima. For the development of these vascular diseases, VSMCs and ECs' inflammatory responses to injury, differentiation, proliferation, migration, and apoptosis are essential cellular events. These complex diseases also involve blood cell recruitment, infiltration, activation, and differentiation. Numerous new molecules have been examined as potential clinical therapies for vascular diseases, which have been the subject of extensive research. In the biology of vascular diseases, the roles of miRs have gradually received more attention in recent years. Over 400 studies have found a link between cardiovascular diseases and altered miR expression profiles. Although vascular remodeling, inflammation, and diseases are all regulated by miRs in several review articles [10], the specific role of miRs in controlling atherosclerosis and restenosis is barely mentioned. Consequently, this audit centers around the jobs of miRs in various kinds of vascular cells according to atherosclerosis and restenosis.

## Conclusion

In both developed and developing nations, atherosclerosis is a prevalent condition with high morbidity and mortality rates. Patients typically die at a high rate from its complications, such as unstable coronary syndromes, myocardial infarction, and stroke. For clinical therapy, a number of drugs and surgical procedures have been used. Angioplasty with stent replacement is typically used to treat atherosclerotic disease patients. Restenosis, on the other hand, is frequently observed in angioplasty patients. Complicated pathophysiological processes highlight both diseases, and extensive research on cellular mechanisms has been conducted to identify therapeutic potential. Over 400 studies have demonstrated the significant roles and functions of MiRs in vascular biology, a novel class of gene regulators. The current understanding of the roles that miRs play in restenosis and atherosclerosis is summarized in this review. Both vascular disorders are caused by ECs, VSMCs, and blood cells. ECs exhibit an inflammatory response, angiogenesis, and migration, with each cell type playing a distinct role in these two conditions; VSMCs that are undergoing differentiation and growth; and blood cells that regulate lipid metabolism and oxLDL uptake. To better understand how miRs alter these cellular functions, we therefore concentrate on the distinct characteristics of each kind of cell. We talked about how angioplasty in animal models and atherosclerotic human specimens alter miR expression profiles significantly. The significance of miRs in the pathogenic processes of vascular diseases is emphasized by these profiles, which provided new insight into the potential clinical applications of miRs. Some miRs, like 126, 1792a, 145, 21, and 146a, have been found to be altered in both in vitro and in vivo studies. There are some miRs that can only be expressed in certain cells or tissues with special status. During embryonic development and in mature vessels, the EC-specific miR-126 and VSMC-specific miR-145 are usually enriched in blood vessels. These miRs are thought to be involved in the development of blood vessels or the maintenance of homeostasis. Through negative modulation of cell cycle regulation as well as of PTEN and p27, miR-21 and miR-221/222 have been investigated as promoters of the proliferation of VSMCs. Due to its high expression levels in various cancer cell lines, MiR-21 is also known as an oncomir. This suggests that these miRs contribute to the pathogenesis of vascular disease. Some miRs are expressed in more than one cell, like miR-146a and miR-155, which are expressed in both ECs and blood cells to trigger an inflammatory response and protect blood vessels, respectively. This suggests that miRs may have tremendous therapeutic potential. Strangely, ongoing advances have empowered the ID of miRs delivered into coursing blood from harmed tissue or profoundly communicated in patients with cardiovascular sicknesses. As a result, circulating miRs and tissue/cell-specific miRs may serve as clinical diagnostic biomarkers for cardiovascular disease patients. The entire body of evidence indicates that miRs may represent novel biomarkers and new therapeutic targets for cardiovascular diseases and have emerged as a new layer of complexity in vascular diseases.

## Acknowledgement

None

## Conflict of Interest

None

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